

## AV junctional versus left atrial rhythm

Nabil El-Sherif

From Cardiology Department, Faculty of Medicine, Cairo University, Egypt, U.A.R.

*Analysis of the scalar configuration and spatial orientation of P waves retrogradely conducted in the junctional tissue in 17 cases of arrhythmias of the QRS-P type and in a case of atrial reciprocal rhythm revealed a consistent retrograde atrial activation in the frontal plane, while the pathway of the atrial depolarization in the horizontal and sagittal planes showed conspicuous variation. In 14 cases (78%) the atrial activation vector was directed from left to right, in 10 cases (55%) it showed also an anterior orientation. These findings represent unequivocal evidence against the theoretical considerations upon which the subject of left atrial rhythm was built.*

*The results of the study suggest that many of the so-called left atrial rhythms may represent in fact instances of junctional rhythm of the P-QRS type (upper nodal rhythm). The work presents sufficient argument against the use of P wave configuration from surface leads as a clue for the site of origin of the impulse or the pathways of intra-atrial conduction.*

In contrast to the extensive analysis and sufficient consistency of the spatial orientation and conventional scalar configuration of P waves of sinus origin, little is available about P waves of AV junctional rhythm. In fact apart from their frequent retrograde activation of the atria – the diaphragmatic surface leads being mostly utilized for their registration – little is known about their spatial orientation and scalar configuration in other planes and other leads, especially the chest leads. The subject acquires significance in view of the revived interest in so-called left atrial rhythm. In spite of the limited capacity of the left atrium to serve as a pacemaker of the heart (Rothberger and Sachs, 1939), automatic cells were described in the left atrioventricular ring and at the pulmonary veno-atrial junctions (Robb and Petri, 1961; Hoffman and Cranefield, 1964), and their spontaneous activity underlies the basis of presumed left atrial rhythms. Relatively little interest was attached to the subject until Mirowski popularized the term on purely theoretical arguments (Mirowski, Neill, and Taussig, 1963). In later publications Mirowski extended his criteria to include variable configurations of the P wave in most of the conventional leads with the exception of lead V6 (Mirowski, 1966a, 1967). The inversion of the P wave in this lead was offered as mandatory and as the most specific feature of left atrial rhythm, being sufficient *per se* to exclude

the possibility of coronary sinus or junctional rhythms (Mirowski, 1966a). Later on Mirowski extended his presumptions for the diagnosis of tachycardias (Mirowski, 1966b) and flutter (Mirowski and Alkan, 1967) of left atrial origin. Shortly after Mirowski published his works it was realized that his theories had at least three limitations. Firstly, the simplified left-to-right relation of the two atria is not in accord with the actual topography of the two chambers (Walmsley and Watson, 1966). Secondly, underlying the concept of the spatial orientation of the left atrial P waves is the assumption that the atrial impulse propagates concentrically through the myocardium in spite of increasing evidence to the contrary (James, 1963; Wagner *et al.*, 1966; Brody, Woolsey, and Arzbacher, 1967). Thirdly, the analysis of the conventional electrocardiogram has the limitations inherent in a non-corrected lead system (Frankl and Soloff, 1968).

Experimentation in the subject of left atrial rhythm usually took the form of analysis of either mechanically induced (Somlyo and Grayzel, 1963) or electrically induced (Massumi and Tawakkol, 1967; Harris *et al.*, 1968) left atrial P waves. The same type of experiment was also frequently used for the study of coronary sinus rhythm (Lancaster *et al.*, 1965; Moore *et al.*, 1967; Massumi and Tawakkol, 1967). In spite of the occasional contradictory conclusions, these experiments helped to cast doubt not only on the consistency of the criteria used for the diagnosis

of left atrial rhythm but also on the use of P wave configuration from surface leads to study the site of origin of the impulse and the pathways of intra-atrial conduction. On the other hand, it was sufficiently clear that the artificial induction of left atrial rhythm with the subsequent description of certain P wave characteristics had nothing to do with the proof of the spontaneous occurrence of the particular rhythm. This consideration primarily guided the line of approach in the present work where we endeavoured to study the scalar configuration and the spatial orientation of P waves conducted retrogradely in the junctional tissue using both conventional and orthogonal scalar leads. The term junctional rhythm is used in the present report, as elaborately defined and suggested by Pick and Langendorf (1968). The study aims to present unequivocal arguments against the theoretical considerations upon which the subject of left atrial rhythm was built (Mirowski, 1966a), and suggests at the same time that many of the so-called left atrial rhythms may represent in fact instances of junctional rhythm of the P-QRS type (upper nodal rhythm).

### Subjects and methods

The atrial activation in impulses arising from junctional tissue may either precede, coincide, or follow the ventricular activation, depending on relative forward and retrograde conductivity rather than on the exact site of impulse formation in the junctional tissue (Pick and Langendorf, 1968). This may lead to the inscription of rhythms of the P-QRS, P/QRS, and QRS-P types (corresponding to the conventional terms of upper, middle, and lower nodal rhythms, respectively). However, in the presence of ectopic rhythms of the P-QRS type, speculations may arise as regards the site of origin of the impulse whether in the junctional tissue, neighbourhood of the coronary sinus, left atrial regions, or other ectopic atrial sites. Study of rhythms of the P/QRS type – as exemplified by cases of isorhythmic AV dissociation with synchronization – suffers from two limitations. Firstly, localization of the site of origin of the P wave, whether secondary to a sinus or junctional impulse, may be extremely difficult in the absence of oesophageal or intra-atrial leads. In fact it has been recently shown that in many of these cases the P waves may prove to have a junctional origin (Waldo *et al.*, 1968). Secondly, and perhaps more specifically related to the present study, is the fact that even if the junctional origin of the P wave can be proved beyond doubt, it will be impossible to analyse its scalar configuration which is partially or completely fused with the QRS complex. Contrary to the above two types, rhythms of the QRS-P type may satisfy – in certain instances – both criteria pertinent to the present study, viz. the unequivocal origin of the P waves whether arising from or traversing the

junctional tissue, and the accessibility for analysis of the scalar configuration and spatial orientation of the atrial complex. Though in many cases of rhythms of the QRS-P type the analysis of the P wave configuration is difficult or impossible because of the superimposition of the P on the T wave, yet in certain instances this may be possible with sufficient accuracy. In a few of these cases the retrogradely conducted P waves may be inscribed clear from the T waves of the preceding ventricular complexes due to the presence of a sufficiently prolonged RP interval. This may be seen in cases of junctional or ventricular tachycardias showing the reversed Wenckebach phenomenon (El-Sherif, El-Ramly, and Sorour, 1969), in cases of ventricular premature systoles showing a second order (delayed) of ventriculo-atrial conduction (Kistin, 1963), and in a few other circumstances.

The P waves in 17 cases of rhythms of the QRS-P type and one case of atrial reciprocal rhythm found to satisfy the above two criteria were subjected to analysis using either the three conventional leads aVF, V<sub>1</sub>, and V<sub>6</sub> (9 cases) or the three orthogonal leads Y, Z, and X (9 cases). In instances of ventricular tachycardia and/or ventricular premature systoles the three conventional or orthogonal scalar leads were recorded simultaneously usually together with an oesophageal VE lead. The Frank lead system (Frank, 1956) was used in the recording of the orthogonal leads. The technique for recording and analysis of the orthogonal scalar leads was previously published (El-Sherif *et al.*, 1966). All records were made on either a 3- or 6-channel Phillips Cardiopan apparatus or a 4-channel Elema-Schonander Mingograph type 42-B. The mean manifest axis angles of the P wave for the three planes of the body (FAP, HAP, and SAP for the frontal, horizontal, and right sagittal planes, respectively) were calculated from the orthogonal scalar leads utilizing the net areas enclosed by the atrial deflections which were enlarged by projection on a screen.

The cases aimed to show the various circumstances under which an unequivocal origin of the P waves, whether arising from or traversing the junctional tissue, can be diagnosed with certainty, and can be grouped under four categories.

A) Thirteen cases of ventricular tachycardia and/or ventricular premature systoles showing retrograde atrial activation, 11 of which were induced during either right ventricular catheterization (9 instances) or left ventricular catheterization (2 instances). In these cases the impulse has to traverse the junctional tissue before giving rise to atrial activation. Seven cases had relatively short RP intervals so that the retrograde P waves fell in the ST segment of the ventricular complex; however, these were always sufficiently clear for analysis (Fig. 1 and 2). In the remaining 6 cases there were instances when a long RP interval was sufficient to carry the retrograde P waves clear from the T complex making their analysis obviously simple (Fig. 3).

B) This group comprised three cases of spon-

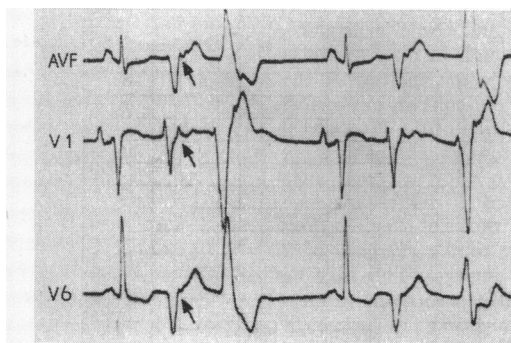


FIG. 1 A study using conventional leads of retrograde atrial activation following ventricular premature systoles. Note that two consecutive ventricular premature systoles follow every sinus beat. All four premature systoles in the figure show ventriculo-atrial conduction. However, the retrograde P waves following the first of the two consecutive premature systoles can be more easily analysed, being negative in aVF, positive in V1, and slightly negative in V6.

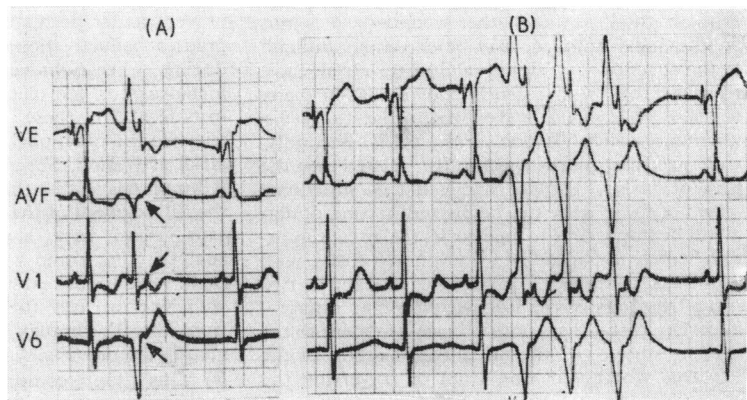
taneous ectopic rhythms of the QRS-P type (lower nodal rhythm) (Fig. 4). Here, a junctional origin of the impulse which meets a sufficient delay in the pathway of retrograde atrial activation in comparison to the forward ventricular conduction is postulated. Though the site of origin of these impulses may be above or below the main or middle portion of the node at its junction with the atria or bundle of His, respectively (Scherf and Cohen, 1964), yet the exact site of origin in the junctional tissue is irrelevant to the present thesis. Rhythms arising from different ectopic atrial foci cannot obviously meet a significant delay in the conduction of the impulse to the rest of the atrial muscle in comparison to the forward ventricular conduction to account for a temporal relation of the QRS-P type. Though interatrial or intra-atrial conduction disturbances can occur (Scherf and Cohen, 1964), yet these evidently affect the contour of the P wave and not the P-QRS relation. C) This included one case of supraventricular tachycardia with 2:1 AV block. In a previous report (El-Sherif, 1970) we commented on the extreme difficulty in differentiating cases of ectopic atrial tachycardia with AV block from cases of AV junctional tachycardia with forward block and normal retrograde conduction, simply because of the loss of the fixed temporal relation between the P and QRS, while the contour of the P wave offers little help. We suggest that such a differentiation may be unwarranted, from the clinical point of view, since the aetiology, clinical picture, salient electrocardiographic features, and management are practically the same and favour the collective term of supraventricular tachycardia with AV block for the whole group. However, in a

unique instance the differentiation may prove possible. This case is shown in Fig. 5. The upper record shows a supraventricular tachycardia with 2:1 AV block (atrial rate 185 beats per minute), the P waves are inverted in diaphragmatic surface leads, lead I, and chest leads from V3 to V6, and positive in V1. From the record it is impossible to tell whether the site of origin of the impulse is in the lower atrial or AV junctional regions. However, the fortuitous occurrence of either a 2:1 exit block at the ectopic centre (record V1 (B)) or 4:1 exit block (not shown in the Fig.) enabled the occurrence of 1:1 relation between atrial and ventricular activations. Here the AV junctional origin could be suggested depending on the temporal relation of the P wave immediately after the QRS complex.

It is pertinent to mention here that in cases of 'classical' supraventricular tachycardia of the QRS-P type, a junctional origin of the impulse cannot be unequivocally established. In these cases there is always the problem of relating the ectopic P waves either to the preceding QRS complex (thus representing instances of 'classical' junctional tachycardia of the QRS-P type) or to the following QRS complex (here the arrhythmia may be either of junctional or ectopic atrial origin with conduction disturbance in the pathway of ventricular activation, i.e. supraventricular tachycardia with grade I AV block). (See Fig. 4 in El-Sherif *et al.* (1969) and Fig. 2 in El-Sherif (1970)).

D) This is one case of atrial reciprocal rhythm: the records in Fig. 6 were obtained from a patient

FIG. 2 A study using conventional leads of retrograde atrial activation following either a ventricular premature systole (strip A) or a short run of ventricular tachycardia (strip B). The retrograde P waves show negative deflection in aVF, positive in V1, and slightly negative in V6. VE represents the oesophageal lead.



with Fallot's tetralogy showing complex conduction disturbances in the AV junctional tissue. Instances of normal AV conduction, 2:1 AV block, 2:1 AV block with interference dissociation, and complete AV dissociation were demonstrated. Of interest in the case were instances of aberration of junctional beats in the presence of complete AV dissociation and instances of atrial reciprocal rhythm easily induced by isoprenaline injection. A complete report of the case is the subject of a separate communication. Pertinent to the present study are the instances of atrial reciprocal rhythm. The site and mechanism of atrial reciprocal rhythm are intimately related to disturbances in the AV junctional conduction (a dual pathway) (Kistin, 1965), thus showing a further mechanism where the unequivocal junctional origin of the P wave can be established. Fig. 6B shows a study by multiple conventional leads for the configuration of the atrial reciprocal beats.

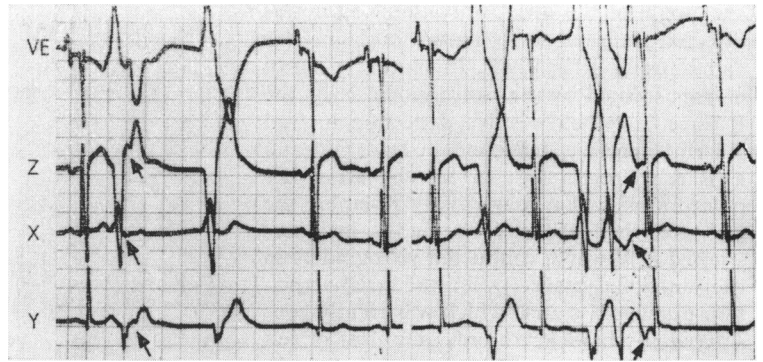


FIG. 3 A study using orthogonal scalar leads for retrograde atrial activation from ventricular premature systoles. This shows 5 ventricular premature systoles. The first one shows retrograde atrial activation with an RP interval of 0.12 sec., which results in the superimposition of the retrograde P waves on the ST-T complex. However, the P waves can still be analysed with reasonable accuracy, being deeply negative in both the Z and Y leads and slightly negative in the X lead. The second and fourth ventricular premature systoles fail to show retrograde atrial activation. The third ventricular premature systole is interpolated between two sinus P waves. There is no retrograde atrial activation; however, concealed retrograde conduction is postulated to explain the slight prolongation of the following sinus PR interval. The fifth ventricular premature systole shows again retrograde atrial activation but with an RP interval of 0.32 sec. The retrograde P waves are now inscribed clear from the T waves of the preceding ventricular complex and can be easily analysed. This prolonged RP interval represents a second order of ventriculoatrial conduction and results in a reciprocal beat, showing a second mechanism for interpolation of ventricular premature systoles (Kistin, 1963). The recordings on the lower channel are not quite simultaneous. VE represents the oesophageal lead.

## Results

Tables 1 and 2 include the analysis of P waves conducted retrogradely in the junctional tissue. While the P waves were consistently inverted in the vertical lead (the conventional lead aVF and the orthogonal Y lead), they showed variable configurations in other leads. The P wave was negative in 7 cases out of 9 in lead V6 and in another 7 cases out of 9 in the orthogonal X lead. In the remaining cases the P wave was either low positive or nearly isoelectric. The P wave in lead V1 showed either positive (5 cases), negative (2 cases), or an initially negative followed by a terminal positive deflection (2 cases). On the other hand, the P wave in the orthogonal Z lead inscribed either a positive (5 cases), negative (3 cases), or an initially negative followed by a terminal positive deflection (1 case). In 10 instances of the present series (half of them studied by the orthogonal leads) the P waves showed a positive deflection in either the conventional lead V1 or the orthogonal Z lead while they inscribed a negative deflection in either the conventional lead V6 or the orthogonal X lead, respectively.

The mean manifest axis angle of the P wave varied within a narrow range in the frontal plane while it showed a wider range of variation in both the horizontal and sagittal planes (Table 2 and Fig. 7). This simply reflected the findings in the scalar configuration of the P wave which was consistently negative in the vertical lead while showing variable contours in both horizontal and sagittal leads.

## Discussion

Analysis of the scalar configuration and spatial orientation of the P waves retrogradely conducted in the junctional tissue revealed a consistent retrograde atrial activation in the frontal plane, while the pathway of the atrial depolarization in the horizontal and sagittal planes showed considerable variation. In 5 out of 9 cases studied by the orthogonal leads, the spatial orientation of the mean manifest

axis angle of the atrial activation vector was directed from left to right with both anterior and superior inclinations. In another 5 out of 9 cases studied by the conventional leads the P wave inscribed positive deflection in lead V<sub>1</sub> and negative deflection in leads V<sub>6</sub> and aVF reflecting a pattern for the atrial depolarization which may closely resemble the one described above. Thus in 10 instances studied of P waves conducted retrogradely in the junctional tissue (55%), the atrial activation pursued a course which was rigidly considered to characterize only impulses of left atrial origin (Mirowski, 1966a). In an additional 4 cases the P waves were negative in the horizontal lead (the conventional lead V<sub>6</sub> or the orthogonal X lead) reflecting a left-to-right atrial activation vector. These findings represent unequivocal evidence against the theoretical considerations upon which the subject of left atrial rhythm was built.

Explanations for the genesis of the pattern of atrial depolarization when the impulse arises from or traverses the junctional tissue are considered outside the scope of this clinical presentation. However, two considerations seem indispensable in this connexion. Firstly, the topography of the left atrium is as posterior to the right atrium as it is to its left side (Walmsley and Watson, 1966). Secondly, the recent elegant demonstrations of specialized conducting pathways within the atrium suggest that a rapid non-concentric propagation of the impulse can occur (James, 1963; Wagner *et al.*, 1966). This contrasts with the conventional concentric propagation upon which all analysis of normal and abnormal P waves is built. Related to the last point the changes in inter- and intra-atrial conduction may play a role (Scherf and Cohen, 1964). These considerations certainly entail the re-evaluation of the whole concept of atrial depolarization.

An important observation in the present series was the consistent retrograde atrial activation in the frontal plane. Review of experimental studies related to the subject showed controversial results. Though most experimental evidence indicates that P waves of junctional or coronary sinus origin are negative in diaphragmatic surface leads (Scherf and Cohen, 1964; Lancaster *et al.*, 1965), some investigators have presented contradictory evidence (Massumi and Tawakkol, 1967; Moore *et al.*, 1967). The reason for the discrepancy between the findings in the present series and some of the experimental reports may be suggested by assuming that the impulse in our group of cases is traversing a more consistent route via junctional tissue

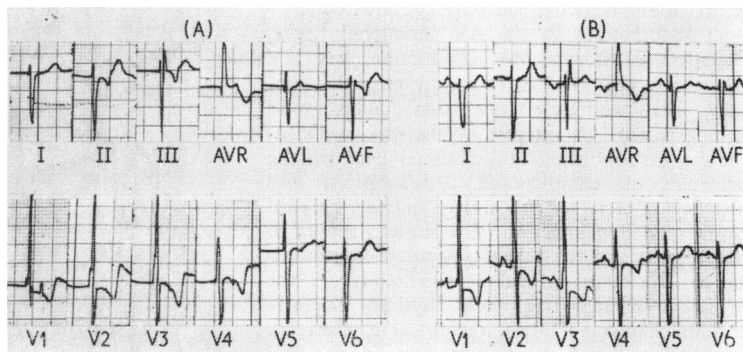


FIG. 4 The electrocardiogram under (A) was recorded from a patient with Fallot's tetralogy and proved normal atrial relations. There is a junctional rhythm of the QRS-P type. The P waves are negative in diaphragmatic surface leads and chest leads from V<sub>3</sub> to V<sub>6</sub>. The P wave in V<sub>1</sub> shows a small initial negativity and a terminal positive deflection. The electrocardiogram under (B) shows establishment of sinus rhythm obtained by intravenous injection of 1 mg. atropine.

FIG. 5 Record (A) shows junctional tachycardia with 2:1 AV block. The P waves show negative deflections in diaphragmatic surface leads, lead I, and chest leads from V<sub>3</sub> to V<sub>6</sub>, and positive in V<sub>1</sub>. The proof that this arrhythmia arises from junctional and not low atrial sites was obtained during the fortuitous occurrence of 2:1 exit block at the ectopic centre (record V<sub>1</sub> (B)) giving rise to 1:1 AV conduction where the temporal relation of the P wave immediately following the QRS complex satisfies the diagnosis of a junctional origin of the tachycardia. The slight difference in the rate of discharge of the ectopic centre during instances of 1:1 conduction at the centre and 2:1 exit block, respectively, can be explained by local changes in the rhythmicity of the ectopic pacemaker.

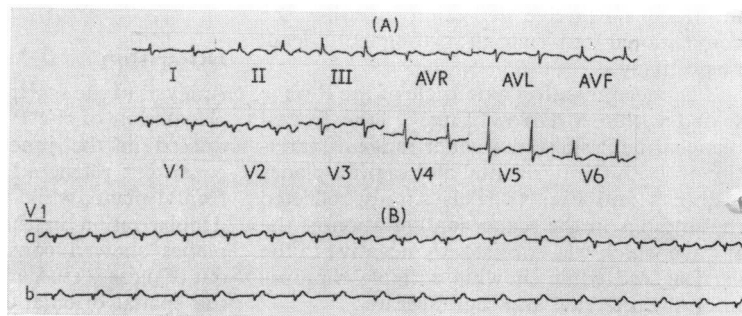
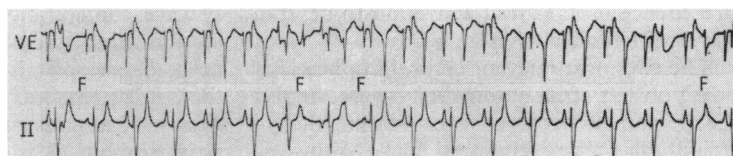
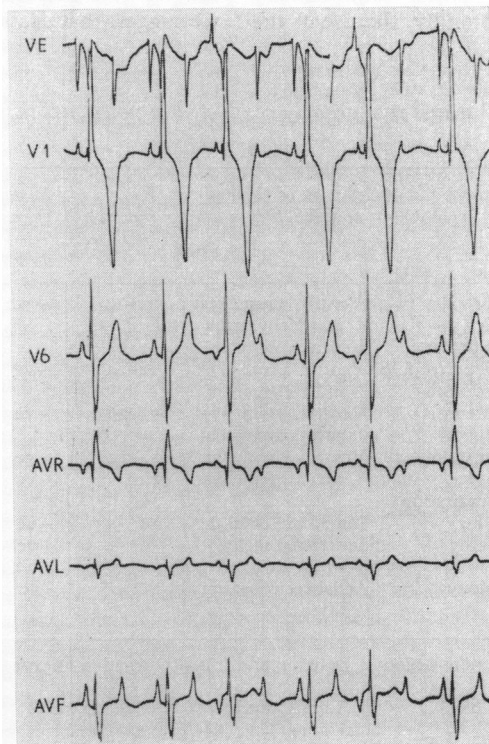


FIG. 6 Record A shows instances of atrial reciprocal beats obtained from a patient with Fallot's tetralogy showing complex disturbances of the AV junctional pathway. The record was obtained shortly after intravenous injection of 0.1 mg. isoprenaline and shows essentially a 2:1 AV block. In addition to the sinus P waves (high positive and peaked deflection in lead II), two other types of P waves are demonstrated. The P waves showing negative deflection represent atrial reciprocal beats, while the P waves with intermediate configuration (marked F) are atrial fusion beats resulting from the simultaneous activation of the atria by both the sinus discharge and the atrial reciprocal beat. Note the 3 consecutive P waves in the middle of the record making a short run of atrial reciprocal rhythm (actually the second P wave in the series is an atrial fusion beat).



(Fig. 6A)



(Fig. 6B)

Record B represents a study by multiple conventional leads for the scalar configuration of the atrial reciprocal beats (the fifth and the ninth P waves). These inscribe a negative deflection in leads V6 and aVF, a positive deflection in leads aVR and aVL, and an rsR' (mainly positive) deflection in lead V1. The standardization is 2.5 times the normal standard in leads V1 and V6 and double the normal standard in lead aVF.

to activate the atria retrogradely. This may be superficially simulated to the consistent route the impulse pursues in sinus rhythm. On the other hand, the impulse in some of the experimental reports may be arising from different sites and is liable to take variable routes. The mechanism by which P waves of junctional and ectopic low atrial origin can inscribe a positive deflection in diaphragmatic surface leads has been based on the assumption of an initial rapid (almost silent) retrograde conduction via the specialized intra-atrial tracts before depolarization of the two atria in the usual downward fashion. Experimental evidence for such a mechanism was previously reported (Brumlik, 1958). The possibility that the site of origin of the ectopic impulse in junctional or atrial sites in close proximity to the pathways of specialized intra-atrial tracts may primarily determine the pattern of atrial activation is only conjectural at the present state of knowledge.

Though vectorcardiographic records using corrected orthogonal leads were sometimes used in the study of the question of left atrial P waves to obviate the limitations inherent in the conventional leads (Frankl and Soloff, 1968; Alfenito, Kennedy, and Varriale, 1969),

yet these studies are subject to the same arguments in the choice of the cases as already discussed for Mirowski electrocardiograms. There is no proof that these studies dealt with left atrial and not junctional rhythms. In fact some of these studies after criticizing Mirowski 'vectorial way of analysis of the conventional leads' have actually suggested junctional origin of their cases (Frankl and Soloff, 1968).

In a recent report, Massumi *et al.* (1969) used the time sequence of right and left atrial depolarization as a guide to the origin of the P waves (using a simultaneous recording of a direct right atrial and a bipolar oesophageal lead). Though the authors were not primarily concerned with the spatial orientation of the P waves, as is this work, yet their findings agree with the views presented in this study. In fact no case of spontaneous ectopic rhythm of the P-QRS type had shown sufficient



precedence of left atrial activation to deserve the term left atrial rhythm.

The two most relevant clinical implications based on left atrial automaticity were, firstly, the presumed occurrence of ectopic tachyarrhythmias from left atrial foci (Mirowski, 1966b, 1967). In fact it has been suggested that atrial flutter represents the chief clinical manifestation of left atrial automaticity (Harris *et al.*, 1968). Secondly, the use of the

P wave configuration as a clinical index for atrial relation in certain instances of congenital heart diseases. It is interesting to note that this same point was the underlying factor for Mirowski to construct his notions about left atrial rhythm (Mirowski *et al.*, 1963). This work presents sufficient evidence against the first point. As regards the second point, which may be of a more direct clinical application, we suggest that caution should be exercised in

TABLE 1 Analysis of P waves retrogradely conducted in junctional tissue using conventional scalar leads

Case no.	Age (yr.) and sex	Clinical diagnosis	Nature of rhythm of QRS-P type	RP interval (0.01 sec.)	Scalar configuration of retrograde P waves		
					aVF	VI	V6
1	23 M	Bilharzial cor pulmonale	Ventric. prem. syst., and tachycardia	11-14	-ve	-ve	Isoelectric
2	16 F	Atrial septal defect	Ventric. prem. syst.	12-24	-ve	+ve	-ve
3	21 M	Idiopathic hypertrophic subaortic stenosis	Ventric. prem. syst., and tachycardia	12-14	-ve	-ve +ve	Low +ve
4	14 M	Pulmonary stenosis	Ventric. prem. syst., and tachycardia	11-26	-ve	-ve	-ve
5	16 M	Bilharzial cor pulmonale	Ventric. prem. syst.	12-14	-ve	+ve	-ve
6	18 M	Mitral stenosis, mitral regurgitation	Ventric. prem. syst.	12	-ve	+ve	-ve
7	5 M	Fallot's tetralogy	Junctional rhythm	14	-ve	-ve +ve	-ve
8	37 M	Cardiomyopathy	Junctional tachy. with exit block	9*	-ve	+ve	-ve
9	25 M	Fallot's tetralogy	Atrial reciprocal rhythm		-ve	+ve†	-ve

-ve, +ve, and -ve +ve denote negative, positive, and initially negative followed by terminal positive deflections, respectively.

\* During periods of 2:1 exit block.

† An rsR configuration of the P wave.

TABLE 2 Analysis of P waves retrogradely conducted in the junctional tissue using orthogonal scalar leads

Case no.	Age (yr.) and sex	Clinical diagnosis	Nature of rhythm of QRS-P type	RP interval (0.01 sec.)	Scalar configuration of retrograde P waves			Mean manifest axis angle of P wave		
					Z	X	Y	Frontal (degrees)	Horizontal (degrees)	Right sagittal (degrees)
1	18 M	Mitral stenosis	Ventric. prem. syst., and tachycardia	14-36	+ve	-ve	-ve	235	112	332
2	22 F	Atrial septal defect	Ventric. prem. syst., and tachycardia	12-34	-ve	Isoelectric	-ve	270	270	218
3	12 F	Patent ductus arteriosus	Ventric. prem. syst.	14	+ve	-ve	-ve	243	117	315
4	25 M	Bilharzial cor pulmonale	Ventric. prem. syst., and tachycardia	12	+ve	-ve	-ve	252	102	328
5	10 M	Fallot's tetralogy	Ventric. prem. syst., and tachycardia	11-14	-ve	-ve	-ve	252	243	237
6	18 M	Congenital aortic stenosis	Ventric. prem. syst.	12-32	-ve	-ve	-ve	255	250	233
7	42 M	Coronary heart disease	Ventric. prem. syst.	14-32	+ve	-ve	-ve	255	108	307
8	17 M	Mitral stenosis, mitral regurgitation	Junctional rhythm	12	+ve	-ve	-ve	225	129	321
9	31 M	Mitral valvotomy	Junctional rhythm	14	-ve +ve	Low +ve	-ve	283	27	277

-ve, +ve, and -ve +ve denote negative, positive, and initially negative followed by terminal positive deflections, respectively.

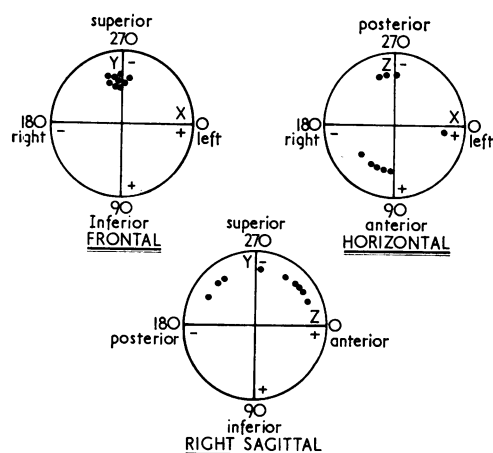


FIG. 7 Spatial distribution of the mean manifest axis angle of 9 cases of P waves conducted retrogradely in the junctional tissue studied by orthogonal scalar leads.

the interpretation of the P wave configuration in the presence of ectopic junctional or atrial rhythms for it is less consistent in comparison with the sinus P wave and may show variable contours. Fortunately enough, despite the variety of circumstances under which junctional rhythm may develop, it occurs relatively infrequently and almost always as a transient phenomenon alternating with sinus rhythm (Friedberg, 1966). We think that in certain circumstances sinus rhythm should be established by amyl nitrite inhalation or atropine injection—in spite of its occasional paradoxical results (Deliyiannis, 1963) — for analysis of the sinus P wave (see Fig. 4).

Though the present study is only peripheral and makes no attempt to suggest a method for the exact localization of the site of ectopic P waves or the pathways of atrial activation, yet it helps to emphasize the limitations inherent in the analysis of records obtained from surface leads — both conventional and orthogonal — in the study of the origin and pathways of ectopic P waves. Probably multiple intracardiac electrodes may prove necessary in this concern. Fortunately enough, these limitations seem at present to be mainly related to P waves of ectopic origin. A sufficient consistency of the scalar configuration of sinus P waves both in health and disease still makes the analysis from surface leads clinically valuable.

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